Supporting Information: Previous studies that reported the relationship between cancers/tumors and genes bold faced in Table 4 or reference proteins used for protein structure prediction

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Citations related to cancer and reference proteins

In the following, we cited references related to each gene one by one. After **Reference Protein**, we denote information about reference proteins.

First comes the name(s) of reference protein(s). Then modelling information comes as follows.

FAMS: P-values computed by blast, aligned region of target protein (total length of target protein), aligned region of reference protein (total length of reference protein), sequence identity.

phyre2: Confidence (max 100), aligned region of target protein (total length of target protein), aligned region of reference protein (total length of reference protein), sequence identity.

ALK

The anaplastic lymphoma kinase (ALK) has been found to be altered in several solid and hematologic tumors [1].

Reference Proteins

ALK was modeled using ABL1 by;

FAMS

[$p = 1 \times 10^{-122}$,969-1394(1620),66-458(465),25%]

and

phyre2

[Conf.100,1107-1394(1620),187-458(465),40%].

Since ABL1 is abbreviation of "Abelson murine leukemia viral oncogene homolog 1", it is oncogenic as its name says.

ASB3

Ankyrin repeat and SOCS box 3 (ASB3) mediates ubiquitination and degradation of tumor necrosis factor receptor II [2].

Reference Proteins

ASB3 was modeled using Ankyrin by;

FAMS

[$p = 1 \times 10^{-104}$, 12-398(518), 2-382(404), 24%]

and

phyre2

[Conf. 100,12-380(518), 2-353(404), 21%].

AVPR1A

AVPR1A is an interesting gene and was investigated from the various points of views. For example, musical aptitude was reported to be associated with AVPR1A-haplotypes [3]. Alternatively, AVPR1A was examined as an autism susceptibility gene [4]. There are no studies that report relationship with cancers.

Reference proteins

AVPR1A was modeled using β2-adrenergic receptor (ADRB2) by;
FAMS
[$p = 1 \times 10^{-102}$, 25-364(418), 137-443(443), 17%]
and
phyre2
[Conf. 100, 47-365(418), 1-441(442), 20%].
There were several studies that report the relationship between ADRB2 and cancers [5]. Preclinical studies have shown that ADRB signalling can inhibit multiple cellular processes involved in breast cancer progression and metastasis, including extracellular matrix invasion, expression of inflammatory and chemotactic cytokines, angiogenesis and tumour immune responses [6]. The ADRB2 and Her2 comprise a positive feedback loop in human breast cancer cells [7].

B3GNT5

B3GNT5 is known to be Lactotriaosylceramide synthase that is related to glycosphingolipid (GPL) synthesis. GPLs were known to be important for embryonic differentiation and development [8,9]. B3GNT5 was involved in seventeen genes that were shown to be differentially expressed between the tumours and the paired normal [10].

Reference proteins

B3GNT5 that was modeled using MFNG by;
FAMS
[$p = 3 \times 10^{-96}$, 89-373(378), 6-240(243), 14%]
and
phyre2
[Conf. 99.9, s 90-365(378), 7-242(243), 23%]
MFNG has also been shown to inhibit Jagged-Notch signalling in cervical cancer cells. Introduction of MFNG into the CaSki cervical cancer cell line leads to inhibition of its tumorigenicity [11].

BCL6

BCL6 is a zinc-finger transcriptional repressor known for its oncogenic role in B cell lymphoma and was expected to be a novel target for therapy of Ph+ B cell acute lymphoblastic leukemia [12]. Inhibitor of BCL6 was reported to kill diffuse large B-cell lymphoma(DLBCL) cells in vitro and in vivo [13]. Other than leukemia, BCL6 was also reported to be expressed in breast cancer and to prevent mammary epithelial differentiation [14].

Reference proteins

BCL6 is in PDB. But, structure was known only partially [5-129 (706)].

BRF1

BRF1, also known as TFIIIB, that was required for transcription initiation at RNA polymerase III promoters consists of subunits, TBP, Bdp1 and Brf1. RNA polymerase (pol) III transcription is specifically elevated in a variety of cancers and is a target of regulation by a variety of tumor suppressors and oncogenes. Deregulation of RNA polymerase III transcription was hypothesized to be mediated by altered TFIIIB expression [15]. In actual, the TFIIIB subunits Brf1 and Brf2 were reported to be differentially expressed in a variety of cancer cell lines [15]. Thus, it is not strange if BRF1 plays critical roles in cancer formation.

Reference proteins

BRF1 was modeled using TFIIB by;
FAMS
[$p = 3 \times 10^{-77}$, 82-281(677), 1-197(207), 21%]
and
phyre2
[Conf, 100, 83-267(677), 2-158(207), 24%]
Overexpression of TFIIB-related factor 2 is significantly correlated with tumor angiogenesis and poor survival in patients with esophageal squamous cell cancer [16].

C1GALT1

Core 1 synthase, glycoprotein-N-acetylgalactosamine 3-beta-galactosyltransferase, 1 (C1GALT1) is known to synthesize O-Glycan (Mucin type O-Glycan biosynthesis). Mucin type O-Glycan biosynthesis is also known to be related to tumor formation as well as development [17, 18].

Reference proteins

C1GALT1 was modeled using MFNG O-Fucosylpeptide 3-Beta-N-Acetylglucosaminyltransferase by;
FAMS
[$p = 1 \times 10^{-113}$, 88-333(363), 6-243(243) 15%]
phyre2
[Conf. 100, 87-329(363), 5-242(243), 18%]
MFNG was also reported to be structurally homologous to B3GNT5 (see above).

CA10

Carbonic anhydrase 10 (CA10) was reported to be one of six genes that were employed as methylation markers for bladder cancer [19].

Reference proteins

CA10 was modeled using other carbonic anhydrase, CA7 by FAMS

[$p = 1 \times 10^{-107}$, 30-303(328), 2-262(262), 29%]

CA1 by phyre2

[Conf. 100, 31-301 (328), 2-258(258), 30%],

Among carbonic anhydrase, CA9 that is the only tumor-associated carbonic anhydrase isoenzyme known [20]. Other than CA9, some carbonic anhydrase were reported to be related to cancer. For example, CA1 was reported to be as a new plasma biomarker for prostate cancer [21]. Carbonic anhydrase inhibitors were recognized as promising antiobesity drugs, too [22].

CAMK1D

Calcium/calmodulin-dependent protein kinase ID (CAMK1D) was found to have subtypes related to severe outcome of breast cancer [23]. CAMK1D SNP was also reported to be associated with BRCA1 and BRCA2 mutation related to breast cancer risk [24].

Reference proteins

CAMK1D is in PDB.

CBLB

CBLB was reported to be one of five genes whose mutation constitute molecular events in chronic myelogenous leukemia [25]. Spontaneous tumor rejection by CBLB-deficient CD8+ T cells was also observed [26]. Thus, CBLB is clearly an important factor for tumor formation.

Reference protein

Although CBLB is in PDB, CBLB was modeled using CBL2 by

FAMS

[$p = 0$, 35-426(982), 1-388(388), 85%];

and

phyre2

[Conf. 100, 39-426(918), 3-388(388), 85%]

CBL2 was suggested to be one of possible candidate genes for familial leukemia with anticipation [27].

CCND1

Cyclin D1 (CCND1) downregulation was known to contributes to anti-cancer effect of isorhapontigenin (ISO) on human bladder cancer cells [28]. CCND1 protein overexpression and CCND1 amplification was observed in breast carcinomas [29]. Amplification of CCND1 was involved in the Wnt and fibroblast growth factor signaling pathways, suggests that these pathways may be activated in esophageal carcinoma cell lines [30]. CCND1 in laryngeal squamous cell carcinoma was reported to have higher expression levels in comparison to healthy controls [31]. MiR-138 suppressed nasopharyngeal carcinoma growth and tumorigenesis by targeting the CCND1 oncogene [32].

Reference proteins

Although CCND1 is in PDB, CCND1 is modeled using ;

CCND3 by FAMS

[$p = 6 \times 10^{-65}$, 24-258(295), 2-229(229), 55%]

and

CDK6 by phyre2

[Conf. 100, 22-257(295), 1-228(233), 20%]

CCND3 was validated as the direct target of miR-138 that was one of 11 down-regulated miRNAs in hepatitis B virus-associated hepatocellular carcinoma [33]. CDK6 was also the direct target of miR-138 that was down-regulated in glioblastoma multiforme [34].

CCNL1

CCNL1 was known to be altered in human head and neck squamous cell carcinoma [35].

Reference protein:

CCNL1 was modeled using cyclin k by;

FAMS

[$p = 9 \times 10^{-91}$, 53-300 (526),7-254 (254), 28%]

and

phyre2

[Conf 100, 48-277(526),2-233(254), 31%].

cyclin k was known to be oncogenic [36].

CD36

CD36 repression was reported to activate a multicellular stromal program shared by high mammographic density and tumor tissues [37]. CD36-mediated activation of endothelial cell apoptosis by an N-terminal recombinant fragment of

thrombospondin-2 inhibits breast cancer growth and metastasis in vivo [38].

Reference proteins

Neither FAMS nor phyre2 could model CD36 with any reliable reference proteins.

CD96

CD96 was known to be a leukemic stem cell-specific marker in human acute myeloid leukemia [39]. Wilms tumor 1 gene mutation-associated gene-expression signature included CD96 [40].

Reference proteins

CD96 was modeled using ;

TTN by FAMS

[$p = 1 \times 10^{-69}$, 23-571(585), 4-493(569),11%]

PIGR by phyre2

[Conf. 99.9,26-486(585),1-441(585), 13%]

Mutations in the gene encoding the giant muscle proteins titin (TTN) have been associated with cardiac and skeletal myopathies in humans [41]. PIGR expression in cancer cells was shown in vitro [42]

CKAP4

Antiproliferative factor decreases Akt phosphorylation and alters gene expression via CKAP4 in T24 bladder carcinoma cells [43].

Reference proteins

CKAP4 was modeled using colicin ia by;

FAMS

[$p = 3 \times 10^{-62}$, 48-428(602), 64-457(602), 9%]

and

phyre2

[Conf. 98.9, 77-516(602), 9-458(602), 10%]

Tumor targeting antibody was generated by mimicing toxin colicin ia [44].

CLYBL

CLYBL was reported to be expressed differently between bladder UCs and UCs and renal pelvis [45].

Reference proteins

CLYBL was modeled using malate synthase by;

FAMS

[$p = 1 \times 10^{-92}$, 3-338 (340), 34-434(501), 13%]

and

phyre2

[Conf. 100, 36-339 (340), 1-324(349), 18%].

Malate synthase catalyse carboxymethyl-forming synthesis of acetyl-CoA while acetyl-CoA carboxylase is known to be a novel target for cancer therapy [46].

CRABP1

CRABP1 was suggested to be included into tumorigenesis of some adenomas subtypes [47]. CRABP1-reduced expression is associated with poorer prognosis in serous and clear cell ovarian adenocarcinoma [48].

Reference proteins

CRABP1 is in PDB.

DNCI1

DNCI1, also termed as DYNC1I1, was reported to be located in Hepatocellular carcinoma amplified region, 7q21.3 [49].

Reference proteins

DNCI1 was modeled using WD-40 repeat by;

FAMS

[$p = 1 \times 10^{-145}$, 75-642(645), 6-569(577), 12%]

and phyre2

[Conf. 100, 200-606(645), 9-577(577), 29%].

Ciao 1, WD40 protein, was reported to interact with the tumor suppressor protein WT1 [50].

EDN2

Forced expression of ET-2 and ET-3 was conducted in human colon cancer cells followed by real-time cell migration and invasion assays [51]. EDN2 were found to be commonly overexpressed in a broad range of human tumour entities [52–54].

Reference proteins

FAMS did not predict reliable structure for EDN2. The structure predicted by phyre2 is too short compared with total length [49-72(178)] and thus useless.

EGLN3

Glioma progression was reported to be suppressed by EGLN3 [55]. EGLN3 was reported to be inactivated by methylation in plasma cell neoplasia [56]. VHL-mutated tumors showed an unexpected overexpression of EGLN3 mRNA [57].

Reference proteins

EGLN3 was modeled using EGLN1 by;

FAMS
[$p = 4 \times 10^{-86}$, 5-227(239),1-222(225),63%]
and
phyre2
[Conf. 100, 9-225(239), 1-216(216), 66%]
EGLN1 was one of twelve newly identified proteins differentially expressed in cancer-associated fibroblasts [58].

EVI1

Overexpression of EVI1 was reported to repress TGF-β signaling in colorectal cancer [59]. Frequent EVI1 translocations in myeloid blast crisis CML that evolves through tyrosine kinase inhibitors [60].

Reference proteins

EVI1 was modeled using
GTF3A by FAMS
[$p = 7 \times 10^{-31}$, 787-978(1051), 4-182(182), 10%]
and
PRDM9 by phyre2
[Conf. 100, 16-212(1051), 4-214(215), 14%]
But the predicted structure is too short compared with total length thus useless.

EXT1

Fibroblast EXT1-levels influence tumor cell proliferation and migration in composite spheroids [61]. Mutation analysis and prenatal diagnosis of EXT1 gene mutations in Chinese patients with multiple osteochondromas [62].

Reference proteins

EXT1 was modeled using EXTL2 by;
FAMS
[$p = 1 \times 10^{-128}$, 478-736(746), 3-265(265), 15%]
and;
phyre2
[Conf. 100, 476-705(746), 1-221(251), 30%]
EXTL2 was a member of tumor suppressor EXT familty together with EXT1 [63].

F2RL3

F2RL3 (PAR4) was often reported to be related to cancer [64–67].

Reference proteins

F2RL3 was modeled using CHRM2 by;
FAMS
[$p = 4 \times 10^{-69}$, 74-355(385), 1-277(278), 19%]
and
phyre2
[Conf. 100, 78-355(385), 5-437(438), 27%]
CCRM2 was hypermethylated in gastric cancer [68].

FADD

FADD protein release mirrors the development and aggressiveness of human non-small cell lung cancer [69]. High levels of phosphorylated FADD (p-FADD) in tumor cells correlate with increased activation of the antiapoptotic transcription factor NF-*kappa*B and is a biomarker for aggressive disease and poor clinical outcome [70].

Reference proteins

FADD was in PDB.

FGF12

Increased FGF12 expression was observed in diffuse type of gastric cancer [71]. Tumour-endothelium interactions in co-culture was reported to elevate FGF12 expression [72]. FGF12 was one of 70 significantly hypermethylated genes in breast cancer tissues [73].

Reference protein

FGF12 was modeled using FGF13 by;
FAMS
[$p = 7 \times 10^{-61}$, 68-216(243), 1-149(149), 76%]
and
phyre2
[Conf. 100, 68-216(243), 1-149(1-149), 77%]
There are no reports that suggest the relationship between FGF13 and cancer/tumor.

FGF19

Endocrine fibroblast growth factor FGF19 promotes prostate cancer progression [74]. Latasa et al suggested that combined targeting of FGF19 and AR/EGFR may enhance therapeutic efficacy [75]. Amplification of FGF19 was involved in the Wnt and fibroblast growth factor signaling pathways, suggests that these pathways may be activated in esophageal carcinoma cell lines [30].

Reference proteins

FGF19 was in PDB. FGF19 was also modeled using other FGF proteins that are widely known to be related to cancer [76].

FGF3

FGF3/FGF4 amplification and multiple lung metastases in responders to sorafenib in hepatocellular carcinoma [77] . Amplification of FGF3 was involved in the Wnt and fibroblast growth factor signaling pathways, suggests that these pathways may be activated in esophageal carcinoma cell lines [30].

Reference proteins

FGF3 was also modeled using various FGF proteins.

FH

Hereditary leiomyomatosis and renal cell carcinoma was characterized by germline mutation of Fumarate Hydratase (FH) [78]. Downregulation of FH is related to tumorigenesis in sporadic renal cell cancer [79].

Reference proteins

FH was in PDB.

GRHL2

Relative expression of a GRHL2-mediated gene-set pair was reported to predict breast cancer metastasis [80]. GRHL2 was also reported to determine the epithelial phenotype of breast cancers and promotes tumor progression [81]. GRHL2 was known to be concordantly deregulated by genomic aberration in gastric cancer [82].

Reference Proteins

FAMS did not predict reliable structure for GRHL2. The structure predicted by phyre2 is too short compared with total length [483-555(625)] and thus useless.

GZMB

EGFR-targeted granzyme B (GZMB) expressed in NK cells enhances natural cytotoxicity and mediates specific killing of tumor cells [83].

Reference Proteins

GZMB is in PDB.

HEBP1

Knockdown of HEBP1, also known as NFE2L2 or NRF2, inhibits the proliferation and growth of U251MG human glioma cells in a mouse xenograft model [84]. HEBP1 prevents initiation but accelerates progression through the Kras signaling pathway during lung carcinogenesis [85].

Reference proteins

FEBP1 was in PDB.

HEY1

HEY1 was reported to be inhibited treatment of mice with EA that inhibits human pancreatic cancer growth in Balb c nude mice [86]. HEY1 was reported to be one of genes down-regulated in SCUBE3-knockdown tumors, while SCUBE3 is strongly expressed in extremely invasive lung carcinoma [87].

Reference proteins

FAMS did not predict reliable structure for HEY1. The structure predicted by phyre2 is too short compared with total length [50-110(304)] and thus useless.

ICA1

ICA1, also known as ICA69, was reported to be a novel Rab2 effector regulating ER-Golgi trafficking in insulinoma cells [88]. ICA1 was mutated in 3 of 9 high-level microsatellite instability (MSI-H) inflammatory bowel disease-related neoplasms vs 2 of 54 sporadic MSI-H colorectal cancers (P = .028) [89].

Reference proteins

ICA1 was modeled using BIN2;

FAMS

[$p = 1 \times 10^{-65}$, 24-259(483), 1-224(230), 13%]

phyre2

[Conf. 99.8, 26-257(483), 3-222(230), 13%]

Bridging integrator-2 (Bin2) is also called Breast cancer-associated protein 1 (BRAP1).

IL1RAP

IL1RAP was reported to be overexpressed on the surface of hematopoietic stem cells of acute myeloid leukemia patients [90]. IL1RAP gene polymorphisms are associated with persistent hepatitis B virus infection [91].

Reference proteins

IL1RAP is in PDB.

IMP-2

IMP2, also known as IGF2BP2, was reported to have tissue-specific expression in colon cancer [92]. IMP2 was also suggested to be a ost-transcriptional drivers of cancer progression [93].

Reference proteins
IMP2 was modeled using
RAVER1 by FAMS
[$p = 5 \times 10^{-38}$, 3-241(599), 23-258(280), 15%]
HNRPL by phyre2
[Conf. 100, 3-158(599), 5-206(209), 18%]
There are no reports that suggest relationship netween RAVER1, HNRPL, and cancer, yet.

IREB2

The minor allele of a variant representing one of the two loci at 15q25 (rs2036534) was associated with increased IREB2 expression in two studies [94].
Reference proteins
IREB2 was modeled using IRP1 by;
FAMS
[$p = 0$, 7-963 (963), 1-887(888), 55%]
and
phyre2
[Conf. 100, 7-963(963),1-887(888), 62 %].
Dysregulation of IRP1-mediated iron metabolism causes gamma ray-specific radioresistance in leukemia cells [95]. IRP1 was reported to be underexpressed in prostate cancer [96].

KCNN3

KCNN3 is also termed as SK3. New alkyl-lipid blockers of SK3 channels was reported to reduce cancer cell migration and occurrence of metastasis [97].
Reference proteins
KCNN3 was modeled using KCNA2 by ;
FAMS
[$p = 1\times10^{-144}$,175-558 (736), 23-388 (389), 14%]
and
phyre2
[Conf. 99.9, 316-558(736), 110-362(363), 20%]
KCNA2, also termed as kv1.2, has not yet any reports that suggest the direct interaction between KCNA2 and cancer. However, KCNA2 is suggested to be related to proliferation [98]. Thus, it is plausible that KCNA2 has potential roles in cancer.

K6HF

Although there were no known reports that suggest relationship K6HF and cancers/tumors, K6HF was known to be related to pachyonychia congenita that is a rare hereditary disorder that can affect the nails, skin, mouth, larynx, hair, and eyes [99].

Reference proteins
K6HF was modeled using bacterial colicin ia by FAMS and phyre2. This may demonstrate that the selection of K6HF as a cancer-related-gene is erroneous.

KLHL6

KLHL6 was known to be mutated in Chronic lymphocytic leukaemia [100]. KLHL6 was reported to be a putative oncogene in Diffuse Large B-cell Lymphoma [101].
Reference proteins
KLHL6 was modeled using KLHL2 by;
FAMS
[$p = 9 \times 10^{-86}$, 321-606(621), 2-286(286), 23%]
and
phyre2
[Conf.100, 318-606(621), 1-286(186), 26%]
KLHL2 (Mayven) was also reported to be related to cancer [102].

LPP

LPP was reported to promote epithelial-to-mesenchymal transition in endometrial carcinomas cooperatively with ETV5 [103]. A complex containing LPP and α-actinin mediates TGFβ-induced migration and invasion of ErbB2-expressing breast cancer cells [104].
Reference proteins
LPP was modeled by FAMS using bacterial protein as reference protein, thus discarded. Phyre2 modeled LPP using LDB1 [Conf. 100, 412-557(612),1-153(157), 28 %]. LDB1 was reported to be essential cofactor to cancer oncogene LMO4 [105].

MAFB

A novel molecular mechanism involved in multiple myeloma development revealed by targeting MAFB to haematopoietic progenitors was proposed [106]. Reexpression of oncoprotein MAFB in proliferative β-cells and Men1 insulinomas in mouse was also reported [107].
Reference proteins
MAFB was in PDB, although only less than half of length [214-303(323)] has predicted structure. Many other MAF family proteins were used as reference proteins.

MCF2L2

MCF2L2 was reported to have higher CNV in adenocarcinoma [108]. CNV in MCF2L2 is even higher in squamous cell carcinoma patients. than in adenocarcinoma patients [109].

Reference proteins

MCF2L2 was modeled using VAV1 by;

FAMS

[$p = 1 \times 10^{-129}$, 431-1005(1114), 2-533(541), 13%]

and

phyre2

[Conf. 100, 612-917(1114), 166-443(541), 21%]

VAV1 was known to fine tune p53 control of apoptosis versus proliferation in breast cancer [110].

MGC26647

There were no known reports that suggest relationship MGC26647, also known as C7orf62, and cancers/tumors,

Reference proteins

MGC26647 was model with appa by;

FAMS

[$p = 6 \times 10^{-26}$, 52-170(253), 5-113(114), 15%]

and

phyre2

[Conf. 98.3, 49-147(253), 2-89(114), 18%]

Although appa is bactrial protein, appa was also known to be homologues to APP [111], amyloid protein precursor, which was known to correlates with early onset of Alzheimer's disease in humans. APP is known to be related to cancer. For example, suppression of APP expression could promote ovarian cancer cell proliferation and invasion [112].

MYEOV

Amplification of MYEOV was involved in the Wnt and fibroblast growth factor signaling pathways, suggests that these pathways may be activated in esophageal carcinoma cell lines [30]. SNP in MYEOV was reported to be relates to prostate cancer risk [113]. Aberrations of MYEOV was reported in neuroblastoma [114].

Reference proteins

Phyre2 and FAMS failed to predict MYEOV protein structure.

NOP5/NOP58

NOP58, also known as nucleolar protein 5, was were down regulated in tumor [115].

Reference proteins

NOP58 was modeled using many of NOP proteins as reference proteins. For example, NOP58 was modeled using NOP56 by ;

FAMS

[$p = 1 \times 10^{-148}$, 28-402(529), 2-366(366), 29%]

and

phyre2

[Conf. 100, 3-399(529),1-350(350), 31%]

NRXN1

Genomic structural variation of NRXN1 was suggested to be related to cancer [116]. MRXN1 was also reported to be one of hepatitis B virus targeted genes [117].

Reference proteins

NRXN1 was in PDB.

NUAK1

High NUAK1, also known as ARK5, expression correlates with poor prognosis and involved in nonsmall cell lung cancer cells migration and invasion [118]. NUAK1 promotes glioma cell invasion, and its elevated expression is correlated with poor clinical outcome [119].

Reference proteins

NUAK1 was modeled using ;

MARK3 by FAMS

[$p = 1 \times 10^{-138}$, 43-370(661), 2-322(322), 40%]

and

PRKCB by phyre2

[Conf. 100, 53-322(661),194-469(520), 28%]

MARKL1, homologous to MARK3, was reported to be elevated in hepatocellular carcinogenesis [120]. PRKCB was reported to be a disease-specific druggable target for treatment of Ewing sarcoma [121].

NXPH1

Median methylation levels of NXPH1 in tumors was $\geq$ 30% higher than in normal samples [122]. NXPH1 was also significantly more likely to be methylated in were significantly more likely to be methylated in IPMNs with high-grade than with low-grade dysplasias with high-grade than with low-grade dysplasia [123].

Reference proteins
NXPH1 was model using;
FLNB by FAMS
[$p = 3 \times 10^{-22}$, 65-192(271), 1-101(108), 12%]
TRIM45 by phyre2
[Conf. 95.5, 120-167(271), 46-95(113), 17%]
FLNB was reported to be a biomarker of hepatocellular carcinoma [124]. TRIM proteins are generally related to cancer [125].

PACS1

PACS1 was reported to be dysregulated in cancer [126].
Reference proteins
FAMS could not find feasible struvutres of PACS1. PACS1 was modeled by phyre2 using yegS [Conf. 96.1, 563-674(963),1-100(265), 17%], but yegS belongs to E. coli, thus there are no reports that suggest the relationship between yegS and cancer.

PDK4

PDK4 was reported to play critical roles in tumor metastasis [127].
Reference proteins
PDK4 was in PDB.

PIGO

PIGO was not reported to be related to cancer.
Reference proteins
PIGO was modeled using ENPP2 by;
FAMS
[$p = 7 \times 10^{-77}$,46-408 (1089), 95-434(805),14%]
and
phyre2
[Conf. 99.9, 52-350(1089), 90-467(783), 23%]
Meta-analysis of 8q24 for seven cancers reveals a locus between NOV and ENPP2 associated with cancer development [128].

PIWIL1

PIWIL1-4 was reported to be significantly higher in tumorous tissue than in adjacent tissue [129].
Reference proteins
PIWIL1 was modeled using AGO2 by;
FAMS (
[$p = 0$, 97-860(861), 2-792(793), 21%]
and
phyre2
[Conf. 100, 99-860(861), 4-792(795), 24%]
There were no studies that report the relationship between AGO2 and cancer. However, PIWIL1 belongs to AGO protein, thus it is natural that AGO2 was used for reference protein.

PKP4

PKP4, also known as p0071, was associated with tumor growth, demonstrating an inverse correlation with tumor size [130]. PKP4 was suggested to have potentials for diagnosis of renal tumours [131].
Reference proteins
PKP4 was modeled using
PG by FAMS
[$p = 1 \times 10^{-109}$, 510-1054(1192), 1-542(546),16%]
PKP1 by phyre2
[Conf. 100, 529-1001(1192), 3-420(420), 37%]
Mutations in the PG and/or changes in its protein levels have been observed in various disease states, including cancer progression or cardiovascular defects [132]. The high specificity of membrane staining for PKP1 was observed in non-small-cell lung cancer [133].

PLA2G4A

Zhang et al [134] found that PLA2G4A were upregulated in gastric cancer. PLA2G4A mutants were reported to modify protective effect of tea consumption against colorectal cancer [135]. Thus, even if PLA2G4A exhibit genotype-specific promoter methylation, it is not strange.
Reference proteins
Structure was reported in PDB.

PLUNC

PLUNC was reported to be an auxiliary tool for the diagnosis of high-grade mucoepidermoid carcinoma of the salivary gland [136].
Reference proteins
PLUNC was modeled using BPI by;
FAMS
[$p = 1 \times 10^{-58}$, 94-245(256), 42-196(217), 13%]
and
phyre2
[Conf. 99.8, 103-238(256), 51-190(217), 16%]

The relation between BPI and inflammatory mediator tumor necrosis factor was reported [137].

PPFIA1

PPFIA1 in laryngeal squamous cell carcinoma was reported to have higher expression levels in comparison to healthy controls [31]. PPFIA1 and CCND1, which was also identified by us, are frequently coamplified in breast cancer [138].

Reference proteins

PPFIA1 was modeled using Liprin-alpha2 (PPFIA2) by ;

FAMS

[$p = 1 \times 10^{-138}$,850-1120 (1202),1-265(265),85%]

and

phyre2

[Conf. 100, 851-1120(1202), 1-261(261), 88%]

PPFIA2 gene was reported to be downregulated by androgens in the human prostate cancer cell line [139].

PTPRT

PTPRT was reported to be tumour suppressor [140].

Reference proteins

PTPRT was modeled using R-PTP-S by;

FAMS

[$p = 0$, 863-1440 (1441), 1-568 (568), 41%]

and

phyre2

[Conf. 100, 863-1440(1441), 1-568(568), 43%]

RPTPS was suggested to play critical roles in human disease including cancer [141].

PVRL3

PVRL3 was identified as Myc-bound loci in medulloblastoma cells [142]. Slight CNV of PVRL3 was observed in renal cell carcinomas [143].

Reference proteins

PVRL3 was in PDB.

RAI17(ZMIZ1)

There are huge number of reports that ZMIZ1 regulate cancers [144, 145]. There are also reports that SNP of ZMIZ1 is associated with cancer [146]. Thus, it is likely that ZMIZ1 exhibits genotype-specific promoter methylaiton in cancer.

Reference proteins

ZMIZ1 was modeled using PIAS2 by;

FAMS

[$p = 8 \times 10^{-86}$, 603-824(1067), 58-261(261), 26%]

and

phyre2

[Conf. 100, 563-824(1067), 6-261(261), 29 %]

Similarity of ZMIZ1 with PIAS protein was already pointed out [147, 148].

RAP2B

RAP2B is overexpressed in many types of tumors [149, 150].

Reference proteins

RAP2B was modeled using RAP2A by;

FAMS

[$p = 2 \times 10^{-53}$,1-164(183), 1-164(167), 93%]

and

phyre2

[Conf. 100, 1-167(183), 1-167(167), 93 %]

RAP2A was one of twelve genes associated with both HNF1b and prostate cancer risk [151].

RAPGEF5

RAPGEF5 was identified as andidate genes involved in tumorigenesis, displaying a robust correlation between expression and genomic alterations [152]. RAPGEF5 was expressed at higher levels in the responding group to erlotinib therapy [153].

Reference proteins

RAPGEF5 was modeled using RAPGEF4 by

FAMS

[$p = 0$,15-579(580), 392-922(922),40%]

and

phyre2

[Conf. 100, 47-579(580),421-922(922), 47 %]

RAPGEF4 is also known as EPAC2. Anthrax edema toxin inhibits endothelial cell chemotaxis via Epac and Rap1 [154].

RND3

Suppression of RND3 promotes cancer cell proliferation and invasion [155]. RND3 [156, 157] is a multifunctional protein and has several interaction with effectors [158]. It also plays critical roles in cancer formations, too [159, 160]. Since mutation of RND3 effector region was reported to attenuate RhoE-mediated disruption of the actin cytoskeleton,

indicating that RhoE exerts its inhibitory effects on ROCK-I through protein(s) binding to its effector region, it is likely reasonable that genotype-specific promoter methylation exists in RND3. Structure was reported in PDB.

Reference proteins

RND3 was in PDB.

RPL14

RPL14 was reported to be increased in the presence of hepatocellular carcinoma (HCC) [161]. Alteration of RPL14 as reported in in squamous cell carcinomas and preneoplastic lesions of the esophagus [162].

Reference proteins

RPL14 was modeled with bacterial proteins, thus useless.

SMAD3

SMAD3 was reported to be expressive in prostate cancer [163].

Reference proteins

SMAD3 was modeled by SMAD2 by;

FAMS

[$p = 1 \times 10^{-121}$, 223-422(425), 1-200(201), 97%]

and

phyre2

[Conf. 100, 223-422(425), 1-200(201), 97%]

SMAD2 was reported to be tumor suppressor [164].

SAMD12

Genomic rearrangement including SAMD12 was found in breast cancer cell line HCC1954 [165, 166].

Reference proteins

SAMD12 was modeled using

PPFIA2 by FAMS

[$p = 1 \times 10^{-28}$, 46-196(201), 51-207(253), 15%]

and

FLI1 by FAMS

[$p = 5 \times 10^{-24}$, 50-162 (201), 14-126(128), 13%]

For the relationship between PPIFA2 and cancer, see PPIFA1. EWS-FLI-1 expression was reported to trigger a Ewing's Sarcoma initiation program in primary Human mesenchymal stem cells [167]. phyre2 modeled SAMD12 using many sam domains. It is natural since SAMD12 means "SAM Domain-Containing Protein 12".

SEMA3C

SEMA3C is involved in the progression of gastric cancer [168]. The influence of neuropilin-1 silencing on semaphorin 3A and 3C activity was reported in B16(F10) murine melanoma cells [169].

Reference proteins

SEMA3C was modeled using SEMA4D by;

FAMS

[$p = 0$, 25-657(751), 3-611(621),32%]

and

phyre2

[Conf. 100, 24-661(751),2-619(621), 36%]

SEMA4D is tumor suppressor [170].

SEMA3E

SEMA3E inhibits the development of tumors from glioblastoma cells implanted in the cortex of the brain [171].

Reference proteins

SEMA3E was modeled using SEMA4D as reference by

FAMS

[$p = 1 \times 10^{-167}$, 28-668(775), 2-611(621), 31%]

and

phyre2

[Conf. 100, 27-678 (775), 1-621(621), 35%]

SEMA3E and SEMA4D were often investigated together and turned out to have similar outcomes [172, 173]. These two were investigated from the point of tumor progression/suppression. Generally, they play a potential roles in tumor formations.

SENP2

SENP2 regulates hepatocellular carcinoma cell growth by modulating the stability of β-catenin [174]. SENP2 deSUMOylating enzyme reverses SUMO-modification of CHFR tumor suppressor, while SUMOylation negatively regulates the stability of CHFR tumor suppressor [175].

Reference proteins

SENP2 was in PDB.

SFRS10

SFRS10, also known as TRA2B, has protein nuclear levels were elevated in poorly differentiated ($p = 0.044$) and lymph node metastases ($p = 0.003$) cancers [176]. Nuclear SFRS10 was reported to be significant in cervical cancer [177].

Reference proteins

SFRS10 was modeled using PSPC1 by;
FAMS as reference protein

[$p = 2 \times 10^{-30}$, 29-261(288), 27-230(255), 17%]
and
phyre2

[Conf. 99.8, 117-198 (288), 8-83(239) 33%]
Knockdown of lncRNA LSINCT5 over expressed in breast and ovarian cancer reduces PSPC1 [178].

STAT4

Genetic variants in STAT4 confers risk of hepatitis B virus-related hepatocellular carcinoma [179]. STAT proteins are reported to be novel cancer drug targets [180].

Reference proteins

STAT4 was modeled using STAT1 by;
FAMS

[$p = 0$, 2-676(748), 1-652(653), 54%]
and
phyre2

[Conf. 100, 2-677(748),1-653(653), 56%]
STAT1 was known to be tumor promoter [181].

STMN2

STMN2 is a novel target of beta-catenin/TCF-mediated transcription in human hepatoma cells [182]. Kidins220-deficient SH-SY5Y cells and native SH-EP1 cells demonstrate down-regulation of the genes DCX and STMN2 [183]

Reference proteins

STMN2 was modeled using STMN4 by;
FAMS

[$p = 2 \times 10^{-29}$, 39-178(179), 2-135(136),67%]
and
phyre2

[Conf. 99.4, 34-174(179), 2-123(124), 78%]
Importance of STMN4 during neuroblastoma differentiation was reported [184].

STXBP6

STXBP6, also termed as amisyn, was known to be a syntaxin-binding protein that may regulate SNARE complex assembly [185] that play critical roles in membrane fusion. Membrane fusion is known to be related to metastasis [186].

Reference proteins

STXBP6 was modeled using VAMP8 by FAMS;

[$p = 1 \times 10^{-28}$, 81-178(210), 174-280(280), 23%].
and
sec3 by phyre2;

[Conf. 20-167(210), 37-181(185), 16%]
Since VAMP8 is a member of SNARE complex, it is reasonable that STXBP6 was modeled using VAMP8. EXOC1, human homolog of sec3, was reported to be one of six genes mapped in chromosome 4 that showed the greatest number of positive and negative regulation between samples [187].

TGFB2

TGFB2 is famous as potential tumor regulator [188].

Reference proteins

TGFB2 was modeled using TGFB1 by;
FAMS

[$p = 2 \times 10^{-77}$, 21-414(414), 3-343(343), 45%]
and phyre2

[Conf. 100, 20-414(414), 1-342(342), 47 %]
TGFB1 was reported to be clinically important for cancer [189].

TMEM16A

TMEM16A induces MAPK and contributes directly to tumorigenesis and cancer progression [190]. TMEM16A was one of 11 genes in 11q13 region exclusively in patients with cervical Lymph node metastasis [191].

Reference proteins

Neither FAMS nor phyre2 predicted reliable protein structures for TMEM16A.

TOMM7

TOMM7 was upregulated in the cell line of L9981 after transfected with NM23-H1 gene [192].

Reference proteins

Neither FAMS nor phyre2 predicted reliable protein structures for TOMM7.

UBP1

There are no studies that report the relationship with cancer.

Reference protein

UBP1 was modeled using CG3027-PA by;
FAMS

[$p = 1 \times 10^{-143}$, 5-384(384),1-347(348),56%]

and
phyre2
[Conf. 100, 6-384(384),1-380(381), 64%]
Since CG3027 is fly protein, there are no studies that report the relation with cancer.

ZA20D2

There are no studies that report the relationship with cancer.
Reference proteins
ZA20D2 was in PDB.

ZBED2

ZBED2 has no reports that suggest relationship to cancers/tumors.
Reference proteins
ZBED2 was in PDB.

ZBTB20

ZBTB20 was reported to be located in one of two two new susceptibility loci for non-cardia gastric cancer [193].
Reference proteins
There are two many short regions modeled by distinct reference proteins (e.e., POZ domain and Zinc finger proteins) with smaller sequence identity (less than 50%) using either FAMS or phyre2, we cannot identify representative reference proteins that model ZBTB20.

ZFPM2

ZFPM2, also known as FOG2, expression in pediatric ovarian sex cord-stromal tumors replicates embryonal gonadal phenotype [194]. Microdeletions were present in ZFPM2 of adult Wilms' tumor [195].
Reference proteins
ZFPM2 was modeled using;
GTF3A by FAMS
[$p = 2 \times 10^{-29}$, 546-729(1151), 5-172(182) 11%]
ZNF297B by phyre2
[Conf. 99.8, 242-380(1151), 3-109(110), 21%]
The predicted structure is too short compared with total length thus useless.

ZNF138

ZNF138 was reported as a putative candidate genes for developmental and malignant disorders [196].
Reference proteins
ZNF138 was modeled using;
AAAT by FAMS
[$p = 2 \times 10^{-62}$, 91-244(262), 1-151(151), 44%]
and
PRDM9 by phyre2
[Conf. 100, 58-262(262), 4-201(215), 6%]
AAAT was proposed to be a inhibitor for pancreatic carcinoma [197]. PRDM9 was proposed to be a highly specific cancer biomarker genes [198].

ZNF588

ZNF588, also known as ZNF107, was reported to have CNV alternation in complex diseases [199].
Reference proteins
ZNF138 was modeled using
AAAT by FAMS
[$p = 2 \times 10^{-52}$, 85-238(783), 1-151(151) 48%]
and
PRDM9 by phyre2
[Conf. 100, 600-770(783), 1-212(215), 6%]
For the relationship between AAAT, PRDM9 and cancers, see ZNF138.

ZNF639

ZNF639, also known as ZASC1, was reported to increase in recurrent oral carcinoma [200]. ZASC1 regulates murine leukemia virus transcription [201].
Reference proteins
ZNF138 was modeled using
AAAT by FAMS
[$p = 1 \times 10^{-55}$, 242-396(485), 1-151(151), 21%]
and
PRDM9 by phyre2
[Conf. 100, 196-485(485), 1-215(215), 7%]
For the relationship between AAAT, PRDM9 and cancers, see ZNF138.

raptor

RNA interference targeting raptor inhibits proliferation of gastric cancer cells [202]. Raptor was suggested to be related to tumor suppressors [203].
Reference proteins
raptor was modeled using

APAF1 by FAMS

[$p = 9 \times 10^{-97}$,410-1318(1335), 243-1119 (1133), 10%]

DDB1 by phyre2

[Conf. 100, 1018-1331(1335), 87-402(402), 15%]

APAF1 was reported to be a target of miR-221 that is overexpressed in neoplasms [204]. DDB1 was reported to be downregulted in pancreatic carcinoma cells [205].

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